LETTERS TO THE EDITOR

Syphilis prevalence is rapidly decreasing in South Korea

The widespread use of penicillin after the second world war resulted in the dramatic decrease in the incidence of syphilis throughout the world. However, the prevalence of syphilis is rising again in western countries owing to an increased use of drugs.[1] In Korea, evaluation of VDRL-positive rates has been carried out in various population groups since the 1960s by various authors. The results ranged from 7·4% in 1962[2] to 2·0% in 1974.[3] However, because of the variability in the population groups, areas and periods of research, it has been difficult to compare and analyse the findings. We began to evaluate VDRL-positive rates in the late 1970s in similar areas with similar population groups and methods in order to standardise the results for accurate analysis of syphilis prevalence in Korea.

Apparently normal Korean adults over 20 years of age, 17,142 in number, were examined from January to December, 1990. Of the study population, 9,151 (7,063 men, 2,088 women) were blood donors in the Seoul area, 5,309 (3,317 men, 1,992 women) were physical examinees examined at Severance Hospital, Yonsei University, and 2,682 were pregnant women delivered at Severance Hospital, Yonsei University. All of the subjects were screened by the VDRL test. The VDRL titration was performed on VDRL-positive pregnant women and physical examinees. The VDRL tests were done according to the Manual of Tests for Syphilis from Centers for Disease Control (CDC). The results obtained were compared with the results of surveys done by the present author group in the similar population groups in 1977,[4] 1981[5] and 1986.[6]

The VDRL-positive rates of blood donors were 2·3% among 6,220 in 1977, 1·0% among 8,501 in 1981, 0·5% among 6,097 in 1986 and 0·3% among 9,151 in 1990. The positive rates in pregnant women were 0·8% among 2,588 in 1981, 0·6% among 1,883 in 1986 and 0·1% among 2,682 in 1990. In physical examinees, the rates were 2·9% among 3,393 in 1977, 1·5% among 2,753 in 1981, 0·8% among 5,136 in 1986 and 0·8% among 5,309 in 1990. The mean positive rate of all three groups for 1977 was 2·5% and the rate dropped to 1·1% in 1981, to 0·6% in 1986, and to 0·4% in 1990 (table).

The marked decrease in the VDRL-positive rates in blood donors and pregnant women implies that the occurrence of new patients is decreasing as the two groups in 1990 consisted mostly of young people under 40 years of age (89% of the blood donors, 99% of the pregnant women). Although the overall incidence is decreasing, the higher VDRL-positive rates in the physical examinees in 1990 is due to a relatively higher proportion of older persons (37% above 40 years of age) and cumulative effect occurring thereby.

To summarise, the VDRL-positive rates among apparently normal adult Koreans are decreasing rapidly since the late 1970s owing to a decrease in the occurrence of new patients.

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Accepted for publication 31 October 1991.

Table Decreasing VDRL-positive rates in apparently normal Korean adults

	No. positive/No. tested (%)			
Year	*Blood donors*	*Physical examinees*	*Pregnant women*	*Total*
1977	144/6,220 (2·3)	98/3,393 (2·9)	—	242/9,613 (2·5)
1981	88/8,501 (1·0)	42/2,753 (1·5)	20/2.588 (0·8)	150/13,842 (1·1)
1986	33/6,097 (0·5)	40/5,136 (0·8)	11/1,883 (0·6)	84/13,116 (0·6)
1990	28/9,151 (0·3)	43/5,309 (0·8)	4/2,682 (0·1)	75/17,142 (0·4)

Cytomegalovirus and AIDS

Cytomegalovirus (CMV) retinitis causes significant morbidity in patients with the Acquired Immune Deficiency Syndrome (AIDS) and is the most common cause of retinitis in this group, occurring in up to 29% of patients.[1] Untreated CMV retinitis usually relentlessly progresses to retinal necrosis and atrophy. Ganciclovir and phosphonoformate

(foscarnet) are effective in slowing the progress of CMV retinitis;[2 3] however, neither is free from toxicity. Both drugs require life-long intravenous administration and ganciclovir may cause bone marrow suppression, while foscarnet may cause renal impairment.[4] We have retrospectively reviewed our experience of CMV retinitis over the period from January 1986 to August 1990 in order to examine survival trends, rates of relapse and complications of treatment.

CMV retinitis was diagnosed on the basis of fundoscopic examination which showed perivascular haemorrhage, exudates and/or periphlebitis typical of CMV retinitis, in 46 patients (45 were homosexual men and one was a bisexual man). Mean age at diagnosis was 36·4 years. All patients received ganciclovir at an initial dose of 10 mg/kg/day for 1 days, followed by maintenance therapy of 5 mg/kg/day using a central venous Hickman catheter. A fall in haemoglobin to below 8 g/dl, total white blood cell count below 1×10^9/l, or platelet count below 20×10^9/l was regarded as unacceptable toxicity. Foscarnet therapy was introduced either in the event of haematological toxicity to ganciclovir or failure of response to ganciclovir with evidence of progressive retinitis.

Median survival following the diagnosis of CMV retinitis was 7 (range 1 to 29) months and did not change significantly over the study period. In two patients CMV retinitis was the presenting AIDS diagnosis; 44 patients had a previous AIDS defining diagnosis.

Complications arising as a result of treatment were common. Only 12 of the 46 cases studied reported no further deterioration in vision and experienced no complications of treatment. This subgroup had a median survival of five months. Overall, haematological toxicity occurred in 16 patients; in 11 haemoglobin levels fell below 8 g/dl, necessitating discontinuation of ganciclovir treatment, with or without transfusion; three patients developed thrombocytopenia, two as part of a pancytopenia and one as an isolated phenomenon requiring platelet transfusion. All these patients recovered adequate haematological function when ganciclovir was stopped. One patient suffered severe nausea and vomiting after the introduction of ganciclovir and elected to stop treatment. Problems related to venous access occurred in nine patients. Hickman line sepsis occurred in five, in two of these *Staphyloccocus aureus* was isolated as the causal organism and in two *Staphyloccocus epidermidis*; in one patient it was necessary to remove the central venous line and administer ganciclovir via a peripheral cannula. Axillary vein thrombosis occurred in four patients; three were successfully treated by anticoagulation, in one the line was removed. One Hickman line had to be resited due to intolerable discomfort at the original insertion site.

Therapy was changed to foscarnet in nine cases; in four this was because of haematological toxicity and in five because of progressive retinitis. Four of these five patients also experienced toxicity from foscarnet. One patient developed acute renal failure three days after starting therapy, another experienced severe vomiting and hypokalaemia, one developed penile ulceration and one became hypercalcaemic, in all cases it was necessary to stop foscarnet treatment.

In our cohort survival following a diagnosis of CMV retinitis did not improve from 1986 to 1990, despite the introduction of zidovudine and primary and secondary prophylaxis against *Pneumocystis carinii* pneumonia. The median survival of seven months is consistent with data from other studies.[5] Overall, less than one quarter of our patients were treated without complications or progressive disease.

Since the completion of this study a number of changes in clinical practice have been introduced. It has now become standard practice to discontinue zidovudine therapy whilst patients are taking high dose ganciclovir, reducing the potential for drug induced myelotoxicity.[6] The introduction of pre-prepared ganciclovir, with the dose individually tailored to the patient's requirements by the manufacturer removes the need for the patient to draw up his/her own drug and may reduce the risk of Hickman line sepsis. Finally CMV resistant to standard therapy and relapsing disease may now be treated with concomitant ganciclovir and foscarnet, although the success of this therapy is yet to be evaluated.

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Accepted for publication 1 October 1991

Oral sex and recurrent vulvo-vaginal candidiasis

The aetiology of recurrent vulvo-vaginal candidiasis (VVC) is not well elucidated. Predisposing factors such as pregnancy, diabetes mellitus, cortico-steroid therapy, severe debilitation, immunosuppression and even the wearing of restrictive clothing are well recognised.[1] None of these factors, however,